Set	Items	Description
S1	11	BOYDEN
S2	1	MAXBAC
S3	1	MATRIGEL
S4	55	HYDRON
S5	1	SEPHACRYL
S6	0	ALCIAN BLUE
S7	1	ALCIAN
S8	0	DECORIN
S9	0	DERMATAN (W) SULFATE
S10	3	DERMATAN
S11	1	SEPHAROSE
S12	1	PHOSPHORIMAGER
S13	1	ROBOCYCLER
S14	0	HISTOEMBEDDER
S15	0	PEROXOBLOCK
S16	0	PEROXO(W)BLOCK
S17	0	HEMO(W)DE'
S18	0	HEMO(W)DE
S19	1	HEMODE
S20	1	REMOVAWELL
S21	1	IMMULON

09/495,448

DIALOG

Set	Items	Description
S1	646208	FIBROBLAST? ?
S2	2995	INVASION(5N)ASSAY
S3	598214	MATRIGEL OR COLLAGEN OR FIBRIN
S4	324	S1 AND S2 AND S3
S5	118425	INTEGRIN? ?
S6	658969	CHEMOTAXIS OR MIGRATION
S7	110	S4 NOT PY>1996
S8	73	RD (unique items)
S9	44	S8 AND S6
S10	8	S9 AND S5
S11	36	S9 NOT S10
S12	1954	ALPHA(2N)6(2N)BETA(2N)1(2N)INTEGRIN
S13	216	S1 AND S12
S14	1	S13 AND S2
S15	78	S13 NOT PY>1995
S16	45	RD (unique items)

?

10/3,AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09860157 BIOSIS NO.: 199598315075
Inhibition of experimental metastasis of human breast carcinoma cells in athymic nude mice by anti-alpha-5-beta-1 fibronectin receptor integrin **antibodies.**
AUTHOR: Newton Sheila A; Reeves Emily J; Gralnick Harvey; Mohla Suresh; Yamada Kenneth M; Olden Kenneth; Akiyama Steven K(a)
AUTHOR ADDRESS: (a)Lab. Dev. Biol., Natl. Inst. Dent. Res., Building 30, Room 421, Natl. Inst. Health, Bethesda, MD**USA
JOURNAL: International Journal of Oncology 6 (5):p1063-1070 1995
ISSN: 1019-6439
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have investigated the role of the human alpha-5-beta-1 fibronectin receptor **integrin** in experimental metastasis. Treatment of human MDA-MB-231 breast carcinoma cells with monoclonal antibodies specific for alpha-5 or beta-1 **integrin** subunits prior to injection into the tail veins of 7 to 9 week old athymic nude mice significantly decreased the median number of lung colonies that were formed. In contrast, treatment of the cells with monoclonal antibodies specific for the alpha-2 subunit had no significant effect. In vitro, the anti-alpha-5 and the anti-beta-1 monoclonal antibodies both strongly inhibited breast carcinoma cell adhesion to fibronectin, while only the anti-beta-1 monoclonal antibody inhibited adhesion to laminin. In a Boyden chamber **invasion assay** , the anti-beta-1 antibody almost completely inhibited invasion of the breast carcinoma cells through an artificial **Matrigel** basement membrane. The anti-a, monoclonal antibody inhibited in vitro invasion approximately 30%, only if **fibroblast** conditioned medium was present as a chemoattractant. Cell **migration** on fibronectin could be inhibited by both the anti-alpha-5 and the anti-beta-1 monoclonal antibody. These results indicate that the alpha-5,beta-1 **integrin** fibronectin receptor on MDA-MB-231 human breast carcinoma cells plays an important role in experimental hematogenous metastasis and may function in this process by a combination of mechanisms, including tumor cell attachment to fibronectin and fibronectin-directed extravasation of tumor cells into the target organ.

1995

10/3,AB/2 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02969385 Genuine Article#: MT852 Number of References: 46
Title: EFFECTS OF EPIDERMAL GROWTH-FACTOR ON INVASIVENESS THROUGH THE EXTRACELLULAR-MATRIX IN HIGH-METASTATIC AND LOW-METASTATIC CLONES OF RCT SARCOMA IN-VITRO (Abstract Available)
Author(s): YUDOH K; MATSUI H; KANAMORI M; MAEDA A; OHMORI K; TSUJI H
Corporate Source: TOYAMA MED & PHARMACEUT UNIV,FAC MED,DEPT ORTHOPAED SURG,2630 SUGITANI/TOYAMA 93001//JAPAN/
Journal: JAPANESE JOURNAL OF CANCER RESEARCH, 1994, V85, N1 (JAN), P63-71
ISSN: 0910-5050
Language: ENGLISH Document Type: ARTICLE
Abstract: We investigated the invasiveness of tumor cells through the

extracellular matrix and the influence of epidermal growth factor (EGF) on tumor cell invasion using in vitro systems in high-[RCT(+)] and low-metastatic [RCT(-)I clones established from poorly differentiated murine RCT sarcoma in C3H/He mice. In the **invasion assay** using a filter coated with reconstituted basement membrane (**Matrigel**) in a Boyden chamber, RCT(+) cells were more invasive than RCT(-) cells. The attachment of RCT(+) cells to extracellular matrix components and the degradation of type IV **collagen** by the cells were significantly greater than with RCT(-) cells. However, there was no significant difference in the **migration** of cells to the extracellular matrix components between cultured RCT(+) and RCT(-) cells. These findings suggested that the different invasiveness of these clone cells was associated with the difference in the ability of attachment to and degradation of the matrix. The level of laminin receptor expression in RCT(+) cells was about four-fold that in RCT(-) cells and laminin stimulated the type IV collagenolytic activity of RCT(+) cells, suggesting that RCT(+) cell attachment to laminin via laminin receptor on the cell surface induced the production of type IV collagenase by the tumor cells. EGF did not affect the invasiveness of RCT(-) cells. In RCT(+) cells, EGF stimulated the invasiveness through **Matrigel** , the attachment to extracellular matrix components and the degradation of type IV **collagen** through high-affinity EGF receptors (EGFR), with K-d Of PM order, while the **migration** to the matrix was not influenced by EGF. These findings suggest that the stimulatory effect of EGF on invasion is related to the acceleration of cell adhesion, and the degradative cascade of the extracellular matrix and high-affinity EGFRs play an important role in the effect of EGF on in vitro invasiveness in this tumor.

10/3,AB/5 (Item 1 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00411517
METHODS FOR MODIFYING THE BINDING OF CELL ADHESION RECEPTORS.
METHODE FUR DIE ANDERUNG DER BINDUNG EINES ZELLADHASIONREZEPTORS.
PROCEDE DE MODIFICATION DE LA LIAISON DE RECEPTEURS D'ADHESION DE CELLULES.
PATENT ASSIGNEE:
LA JOLLA CANCER RESEARCH FOUNDATION, (443361), 10901 North Torrey Pines Road, La Jolla, CA 92037, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)
INVENTOR:
RUOSLAHTI, Erkki, I., P.O. Box 1054, Rancho Santa Fe, CA 92067, (US)
GAILIT, James, O., 3914 Centre Street, No. 12, San Diego, CA 92103, (US)
GEHLSEN, Kurt, R., 7646-16 Palmilla Drive, San Diego, CA 92122, (US)
HAUTANEN, Aarno, 8655 Via Mallorca, 97, La Jolla, CA 92037, (US)
PIERSCHBACHER, Michael, D., 1111 Alexandria Drive, San Diego, CA 92107, (US)
LEGAL REPRESENTATIVE:
UEXKULL & STOLBERG Patentanwalte (100011), Beselerstrasse 4, W-2000 Hamburg 52, (DE)
PATENT (CC, No, Kind, Date): EP 435946 A1 910710 (Basic)
WO 9002556 900322
APPLICATION (CC, No, Date): EP 89911334 890913; WO 89US3979 890913
PRIORITY (CC, No, Date): US 244701 880914
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: A61K-033/32; A61K-037/12; G01N-033/53; G01N-033/68; C07K-003/20; A61K-037/02
NOTE:
No A-document published by EPO

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	EPBBF2	1618
CLAIMS B	(German)	EPBBF2	905
CLAIMS B	(French)	EPBBF2	1060
SPEC B	(English)	EPBBF2	8389

Total word count - document A 0
Total word count - document B 11972
Total word count - documents A + B 11972
>>>Item 10 is not within valid item range for file 670
?

11/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08893194 95196217 PMID: 7889524
Enhanced effect of epidermal growth factor on pulmonary metastasis and in vitro invasion of rat mammary carcinoma cells.
Hamada J; Nagayasu H; Takayama M; Kawano T; Hosokawa M; Takeichi N
Laboratory of Cell Biology, Hokkaido University School of Medicine, Sappooro, Japan.
Cancer letters (IRELAND) Mar 2 1995, 89 (2) p161-7, ISSN 0304-3835 Journal Code: CMX
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
We examined the effects of epidermal growth factor (EGF) on metastatic and in vitro invasive capacity of weakly malignant ER-1 cells derived from a rat mammary carcinoma cell line, c-SST-2. EGF enhanced the metastatic capacity and in vitro invasiveness to reconstituted basement membrane, **Matrigel** , of ER-1 cells in a dose-dependent fashion. EGF-stimulated invasiveness was inhibited by anti-EGF antibody, which is able to neutralize the binding of EGF to EGF receptor, in the **invasion assay** system. EGF stimulated chemotactic **migration** toward fibronectin, laminin or newborn rat **fibroblast** -conditioned medium which was used as a chemoattractant in the in vitro **invasion assay** , but showed neither adhesion to **Matrigel** nor production of gelatinase and plasminogen activators. These results suggested that the increased metastatic and invasive capacity of ER-1 cells by EGF might be due to the increase in cell motility.

11/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07896016 93265428 PMID: 8495401
Development of a novel human extracellular matrix for quantitation of the invasiveness of human cells.
Siegal GP; Wang MH; Rinehart CA; Kennedy JW; Goodly LJ; Miller Y; Kaufman DG; Singh RK
Department of Pathology, University of Alabama, Birmingham, UAB Comprehensive Cancer Center 35233-1924.
Cancer letters (NETHERLANDS) Apr 30 1993, 69 (2) p123-32, ISSN 0304-3835 Journal Code: CMX
Contract/Grant No.: CA31261, CA, NCI; CA45727, CA, NCI; ES07017, ES, NIEHS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
During the crucial stages of tumor cell invasion and metastasis, neoplastic cells must traverse extracellular matrices for their **migration** to distant sites. Because basement membranes (BM) serve as a critical barrier to such passages, most previous in vitro assay models have utilized either an intact BM or a reconstituted rodent or avian BM-matrix to study this process. We have created a gel-like extracellular matrix derived from human amnions which contained type IV **collagen** , laminin, entactin, tenascin and heparan sulfate proteoglycan. This matrix, which we called Amgel, was used to study selected steps of invasion including cell attachment to matrix, degradation of it by proteolytic enzymes and movement of human tumor cells through matrix defects. An efficient tumor **invasion assay** system was developed utilizing filter-supported uniform coatings of this matrix in chambers. Human tumor cells (HT-1080 fibrosarcoma and RL-95

adenocarcinoma), when seeded onto Amgel-coated membranes, attached to matrix within 2 h and initiated a time-dependent **migration** and invasion process, as verified by biochemical analysis and both light and electron microscopy. In an optimized **invasion assay** 12-15% of tumor cells completely traversed the matrix during a 72-h period with > 90% viability. In contrast to these highly-invasive cells, normal human foreskin **fibroblasts** and normal human endometrial stromal cells exhibited minimal **migration** /matrix penetration during the same time period. When the Amgel-selected tumor cells (i.e. those penetrating the barrier) were isolated, subcultured, and re-exposed to Amgel, they had heightened invasiveness (2-3-fold) as compared to the parental cells. Thus, this improved 'all human' system for quantitating the invasive ability of tumor cells may provide a valuable tool in dissecting out the mechanistic underpinnings of human metastasis. In addition, this assay has the ability to screen agents which have potential anti-invasive and by extension anti-metastatic, activity or chemotactic properties.

11/3,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07755985 93150981 PMID: 1283496
Use of a reconstituted basement membrane to study the invasiveness of tumor cells.
Iwamoto Y; Sugioka Y
Department of Orthopaedic Surgery, Faculty of Medicine, Kyushu University, Fukuoka, Japan.
Advances in experimental medicine and biology (UNITED STATES) 1992, 324 p141-9, ISSN 0065-2598 Journal Code: 2LU
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
We have used an extract of basement membranes which can be reconstituted into a biologically active gel matrix composed predominantly of **collagen** IV, laminin, nidogen, and heparin sulfate proteoglycan, in order to study the mechanisms involved in tumor cell invasion. When layered onto a porous filter in a Boyden chamber, the gel forms a barrier to the passage of normal cells. Malignant cells are able to cross this layer when the conditioned medium of NIH 3T3 cells is used as a chemoattractant to stimulate cell **migration** . A variety of human tumor cells have thus been studied in this system and we find a high correlation between their invasiveness in vitro and their malignant behavior as exhibited in vivo. We have used this in vitro **invasion assay** to test for factors which might inhibit tumor cell invasion. Collagenase IV is produced by malignant cells and is thought to be required for invasion. Indeed, inhibitors of this enzyme have demonstrated reduced tumor cell invasiveness. One site of five amino acids, on the B1 chain, which has been shown to promote cell adhesion, **migration** and binding to laminin receptor, was found to inhibit the invasion of tumor cells. In addition, factors which elevated cAMP levels were also able to suppress the invasiveness of tumor cells. These data suggest that the assay system described herein can be successfully utilized to study the invasive activity of tumor cells and those factors that may inhibit the spread of malignant cells.

11/3,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07720681 93224275 PMID: 1298737
Evaluation of an in vitro invasion assay **for use on solid tissue samples and cultured cells.**

Mackinnon WB; Hancock R; Dyne M; Russell P; Mountford CE
University of Sydney, NSW, Australia.
Invasion & metastasis (SWITZERLAND) 1992, 12 (5-6) p241-52, ISSN 0251-1789 Journal Code: GV4
Contract/Grant No.: CA 51 054-02, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
An **invasion assay** , developed for monitoring the in vitro penetration of reconstituted basement membrane, **matrigel** , was modified and successfully applied to solid tumours, normal tissues as well as a variety of normal and tumour cell lines. However, we found that some normal **fibroblasts** were capable of in vitro invasion whilst some malignant cell lines with invasive capacity in vivo did not penetrate the **matrigel** . Nevertheless, this method can distinguish invasive capacity within a tumour model, and as a consequence may be used to elucidate some of the biochemical mechanisms in the invasion process comparing cells grown both in vitro and in vivo. Since the method does not always correlate with invasion in vivo the results must be interpreted with caution.

11/3,AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07699495 93194483 PMID: 1294530
Evaluation of in vitro reconstituted basement membrane assay to assess the invasiveness of tumor cells.
Simon N; Noel A; Foidart JM
Laboratory of Biology, University of Liege, Belgium.
Invasion & metastasis (SWITZERLAND) 1992, 12 (3-4) p156-67, ISSN 0251-1789 Journal Code: GV4
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The crossing of tumor cells through basement membranes represents a critical step in the metastatic process. We have used a reconstituted basement membrane (**matrigel**) coated on filter in a Boyden chamber to assess the invasive potential of tumor and normal cells. No correlation was found between chemoinvasion in vitro and the metastatic potential in vivo. Normal human **fibroblasts** and murine 3T3 **fibroblasts** penetrated filters coated with **matrigel** . On the other hand, the tumoral cells (MCF7, MCF7 gpt, MCF7 ras, BeWo, JAR, NUC-1 cells) were unable to cross the matrix. Our results suggest that in our conditions, this widely used model to assess tumoral **invasion** does not provide a universal **assay** to test the invasiveness of tumor cells. Penetration of the **matrigel** appears to be related to chemotactic or haptotactic responses depending upon cell types. Our data emphasize the variability of molecular events associated with basement membrane invasion.

11/3,AB/13 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03322345 Genuine Article#: NW289 Number of References: 37
Title: USE OF THE MATRIGEL-**BASED ASSAY TO MEASURE THE INVASIVENESS OF LEUKEMIC-CELLS** (Abstract Available)
Author(s): JANIAK M; HASHMI HR; JANOWSKAWIECZOREK A
Corporate Source: UNIV ALBERTA,DEPT MED,DIV CLIN HEMATOL,8249-114ST/EDMONTON T6G 2R8/AB/CANADA/; UNIV ALBERTA,DEPT MED,DIV CLIN HEMATOL/EDMONTONT6G 2R8/AB/CANADA/; UNIV ALBERTA,CROSS

CANC INST/EDMONTON/AB/CANADA/; CANADIAN RED CROSS,CTR BLOOD/EDMONTON/AB/CANADA/
Journal: EXPERIMENTAL HEMATOLOGY, 1994, V22, N7 (JUL), P559-565
ISSN: 0301-472X
Language: ENGLISH Document Type: ARTICLE
Abstract: The reconstituted basement membrane (**Matrigel**)-based assay was used to quantify the invasive potential of hematopoietic cells including cultured human leukemic cells (KG-1, K-562, HEL, HL-60, and U-937), normal bone marrow (BM) cells, and normal polymorphonuclear leukocytes (PMNL). We found that (i) in contrast to 6- to 72-hour incubation periods typically used in assays with solid tumor cells, most of the invasive cell populations tested here required only 2 to 4 hours to cross the **Matrigel** layer; (ii) unlike that of PMNL, whose invasiveness was stimulated by the addition of FMLP, the invasive rate of cultured leukemic cells was not affected by this chemoattractant; (iii) the rate of invasion was inversely proportional to the **Matrigel** concentration per filter but varied with the **Matrigel** batch used; (iv) the most consistent results were obtained when 2.5 to 4x10(5) cells were added to the top portion of the blind well; and (v) of all leukemic cells tested, the least differentiated myeloblastic KG-1 cells exhibited the highest invasive potential, which was comparable to that of normal PMNL. We conclude that the **Matrigel** -based assay can be used as a model system in studies of mechanisms regulating movement of hematopoietic cells across basement membrane barriers.

11/3,AB/18 (Item 3 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00207049
Reconstituted basement membrane complex with biological activity.
Wiederhergestellter Basalmembrankomplex mit biologischer Aktivitat.
Complexe de membrane basilaire reconstitue a activite biologique.
PATENT ASSIGNEE:
THE UNITED STATES OF AMERICA as represented by the Secretary, United States Department of Commerce, (301900), National Technical Information Service, Office of Government Inventions and Patents, 5285 Port Royal Road, Springfield, Virginia 22161, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)
INVENTOR:
Kleinman, Hynda Karen, 6405 Winston Drive, Bethesda, MD 20817, (US)
Martin, George Reilly, 5507 Charles Street, Bethesda, MD 20817, (US)
LEGAL REPRESENTATIVE:
Luksch, Giorgio, Dr.-Ing. et al (42332), Ing. A. Giambrocono & C. S.r.l. Via Rosolino Pilo, 19/b, I-20129 Milano, (IT)
PATENT (CC, No, Kind, Date): EP 218065 A2 870415 (Basic)
EP 218065 A3 890308
EP 218065 B1 910814
APPLICATION (CC, No, Date): EP 86111635 860822;
PRIORITY (CC, No, Date): US 771409 850830; US 867027 860527
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: C12N-005/00; A61K-035/12; A61K-035/50; C12Q-001/02; C12Q-001/24;

ABSTRACT EP 218065 A2
The present invention discloses a biologically active basement membrane composition. When polymerized under physiological conditions, the composition forms gel-like structures whose ultrastructure resembles interconnected thin sheets of the lamina densa zone of basement membrane. The major components of the composition include laminin, type IV

collagen , heparan sulfate proteoglycan, entactin and nidogen. These components polymerize in constant proportions when redissolved and allowed to reconstitute. Molecular sieve studies on the soluble extract demonstrate that laminin, entactin and nidogen are associated in a large but dissociable complex. The reconstituted matrix is biologically active and stimulates the growth and differentiation of a variety of cells, including epithelial cells, nerve cells, hair follicles and the like. The reconstituted matrix can also be used for determining metastatic potential of tumor cells and for isolating metastatic tumor cells.
ABSTRACT WORD COUNT: 135

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	EPBBF1	939
CLAIMS B	(German)	EPBBF1	717
CLAIMS B	(French)	EPBBF1	770
SPEC B	(English)	EPBBF1	5402
Total word count - document A			0
Total word count - document B			7828
Total word count - documents A + B			7828

11/3,AB/32 (Item 1 from file: 653)
DIALOG(R)File 653:US Patents Fulltext

01767460
Utility
RECONSTITUTED BASEMENT MEMBRANE COMPLEX WITH BIOLOGICAL ACTIVITY
[CELL GROWTH, DIFFERENTIATION; PROTEIN EXTRACT OF LAMININ, **COLLAGEN** , HEPARIN SULFATE GLYCOPROTEIN, ENTACTIN, NIDOGEN]

PATENT NO.: 4,829,000
ISSUED: May 09, 1989 (19890509)
INVENTOR(s): Kleinman, Hynda K., Bethesda, MD (Maryland), US (United States of America)
Martin, George R., Bethesda, MD (Maryland), US (United States of America)
ASSIGNEE(s): The United States of America as represented by the Secretary of the Department of Health and Human Services, (A U.S. Government Agency), Washington, DC (District of Columbia), US (United States of America)
[Assignee Code(s): 6814]
APPL. NO.: 6-867,027
FILED: May 27, 1986 (19860527)

BACKGROUND OF THE INVENTION

This application is a continuation-in-part of U.S. application Ser. No. 06-771,409, filed Aug. 30, 1985, now abandoned.

FULL TEXT: 701 lines

ABSTRACT

The present invention discloses a biologically active basement membrane composition. When polymerized under physiological conditions, the composition forms gel-like structures whose ultrastructure resembles interconnected thin sheets of the lamina densa zone of basement membrane.

The major components of the composition include laminin, type IV **collagen** , heparin sulfate proteoglycan, entactin and nidogen. These components polymerize in constant proportions when redissolved and allowed to reconstitute. Molecular sieve studies on the soluble extract demonstrate that laminin, entactin and nidogen are associated in a large but dissociable complex. The reconstituted matrix is biologically active and stimulates the growth and differentiation of a variety of cells, including epithelial cells, nerve cells, hair follicles and the like. The reconstituted matrix can also be used for determining metastatic potential of tumor cells and for isolating metastatic tumor cells.
?

16/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08743781 96087665 PMID: 8537446
Differential distribution of two cytoplasmic variants of the alpha 6 beta 1 integrin **laminin receptor in the ventral plasma membrane of embryonic** fibroblasts.
Cattelino A; Longhi R; de Curtis I
Department of Biological and Technological Research (DIBIT), S. Raffaele Scientific Institute, Milano, Italy.
Journal of cell science (ENGLAND) Sep 1995, 108 (Pt 9) p3067-78, ISSN 0021-9533 Journal Code: HNK
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The **integrin alpha 6 beta 1** is a receptor involved in the adhesion of several cell types to laminin. By using function-blocking antibodies, we have shown that alpha 6 beta 1 is a functional laminin receptor in chick embryo **fibroblasts** . We also found that these cells express two variants of the alpha 6 subunit, alpha 6A and alpha 6B, characterized by different cytoplasmic domains. By using indirect immunofluorescence with isoform-specific polyclonal antibodies, we showed that the two isoforms of the alpha 6 subunit distribute differently on the ventral plasma membrane of these cells cultured on laminin-coated substrates. In fact, while the alpha 6A subunit was found codistributing with vinculin in focal contacts, the alpha 6B subunit showed a homogeneously distributed punctate pattern. This difference was particularly evident when preparations of ventral plasma membranes were used for the immunolocalization. Furthermore, when cells were cultured on fibronectin, a substrate not recognized by the alpha 6 beta 1 laminin receptor, the distribution of the two alpha 6 isoforms was similar to that observed on laminin, with alpha 6A still colocalizing with vinculin in focal adhesions. Our results indicate that two forms of the alpha 6 beta 1 laminin receptor coexpressed in the same cells show distinctive distributions, and suggest that receptor occupancy by laminin is not essential for the accumulation of the alpha 6A beta 1 integrin in adhesion plaques.

16/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08655858 96068197 PMID: 7479613
Characteristics of trophoblast cells migrating from first trimester chorionic villus explants and propagated in culture.
Irving JA; Lysiak JJ; Graham CH; Hearn S; Han VK; Lala PK
Department of Anatomy, University of Western Ontario, London, Canada.
Placenta (ENGLAND) Jul 1995, 16 (5) p413-33, ISSN 0143-4004 Journal Code: PMN
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
We developed a method of propagating pure first trimester human trophoblast cells growing out of primary explants of mechanically derived chorionic villus fragments (Yagel et al, 1989; Graham et al, 1992). We have now extensively characterized these cells during their initial outgrowth and in long-term culture, employing a variety of markers and techniques as outlined below. By double label immunofluorescence using epithelial (cytokeratin) and mesenchymal (vimentin) cell markers, we identified the chorionic villus migrant cell populations as pure trophoblast (39 per cent of outgrowths) or a mixture of trophoblast and **fibroblast** (61 per cent).

Further phenotyping of the pure trophoblast outgrowths by double label immunostaining using anti-cytokeratin antibody and a panel of other primary antisera revealed that these cells exhibit a variety of markers characteristic of extravillous invasive trophoblast cells in situ: insulin-like growth factor (IGF)-II, NDOG-5, proliferating cell nuclear antigen (PCNA), human leucocyte antigen framework antigen (W6/32) and a distinct set of integrins including alpha 1, alpha 3, alpha 5, alpha v and beta **1** subunits and alpha v **beta** 3/**beta** 5 vitonectin receptor. They were negative for **alpha** **6** and **beta** 4 **integrin** subunits. Immunogold electron microscopy of explants grown on type IV collagen gel revealed the production of conventional and oncofetal types of fibronectin by mononucleate trophoblast cells and human placental lactogen by multinucleate cells. Immunolabelling, flow cytometry and immunoprecipitation revealed that this phenotypic profile was retained with complete fidelity in the long-term culture; thus, trophoblasts migrating out of first trimester chorionic villus explants and their propagated progeny belong to the invasive extravillous trophoblast of the placenta.

16/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08622702 96005842 PMID: 7558139

Polymorphonuclear leucocyte migration through human dermal fibroblast **monolayers is dependent on both beta 2-integrin (CD11/CD18) and beta 1-integrin (CD29) mechanisms.**

Gao JX; Issekutz AC

Department of Pediatrics, Microbiology and Immunology, Dalhousie University, Halifax, Nova Scotia, Canada.

Immunology (ENGLAND) Jul 1995, 85 (3) p485-94, ISSN 0019-2805 Journal Code: GH7

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Accumulation of leucocytes in inflammation involves their migration through vascular endothelium and then in the connective tissue. We investigated human polymorphonuclear leucocyte (PMNL) migration through a biological barrier of human dermal **fibroblasts** grown on microporous filters, as a model of PMNL migration in the connective tissue. PMNL did not migrate through a **fibroblast** monolayer unless a chemotactic factor, e.g. C5a, interleukin-8 (IL-8) or zymosan-activated plasma (ZAP; C5adesArg), was added. This migration was partially inhibited (35-70%, depending on the stimulus) by treatment of PMNL with monoclonal antibody (mAb) to CD18 (beta 2-integrins). Most of the CD18-independent migration was inhibited by mAb to beta 1-integrins (CD29). Inhibition by mAb to beta 1 was observed when the PMNL, but not the **fibroblasts** , were treated with mAb. The role of beta 1-integrins in PMNL transfibroblast migration was detectable only when the function of the CD11-CD18 complex was blocked, because mAb to beta 1-integrin alone had no significant effect on PMNL migration. Migration induced by C5a was more CD18-independent compared to IL-8 or C5adesArg. The CD18-independent migration was also inhibited by mAb to the **beta** **1** **-integrin** subunits **alpha** 5 (of very late antigens-5; VLA-5) and **alpha** **6** (of VLA-**6**). Treatment of the **fibroblasts** (4 hr) with tumour necrosis factor-alpha (TNF-alpha) or IL-1 alpha enhanced C5a-induced PMNL transfibroblast migration and increased the proportion of migration utilizing the CD11-CD18 mechanism. However, TNF-alpha treatment had no effect on the degree of beta 1-integrin-dependent migration. These findings suggest that in response to the chemotactic factors C5a, IL-8 and C5adesArg, PMNL migration in the connective tissue is mediated by both CD11-CD18 (beta 2) and beta 1-integrins on the PMNL. The VLA-5 and VLA-6 members of beta 1-integrins are involved in this process. This is in

contrast to PMNL migration across endothelium in this system, which is virtually all CD18 dependent with no significant role for beta 1-integrins.

16/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08617818 96018119 PMID: 7551560
Divalent cations (Mg2+, Ca2+) differentially influence the beta 1 integrin-mediated migration of human fibroblasts **and keratinocytes to different extracellular matrix proteins.**
Lange TS; Kirchberg J; Bielinsky AK; Leuker A; Bank I; Ruzicka T; Scharffetter-Kochanek K
Department of Dermatology, University of Dusseldorf, Germany.
Experimental dermatology (DENMARK) Jun 1995, 4 (3) p130-7, ISSN 0906-6705 Journal Code: B06
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Directed migration of keratinocytes and **fibroblasts** is a fundamental prerequisite in wound healing. Cation-dependent affinity changes of integrins are responsible for cell adhesion to and deadhesion from extracellular matrix proteins and have been implicated in driving cell migration. The specific requirements for divalent cations in the integrin-dependent migration of human dermal **fibroblasts** and human epidermal keratinocytes to various extracellular matrix proteins have been studied in vitro using blindwell Boyden chambers. The migration of the tested cells to collagen type I was mediated by the alpha 2 **beta 1** integrins, to fibronectin by the combined action of the **alpha** 3 **beta 1** and the **alpha** 5 **beta 1 integrin** , and the migration of **fibroblasts** to laminin dependent both on the **alpha** 2 **beta 1** and the alpha **6 beta 1** integrins. No migration of keratinocytes to laminin was detected. Mg2+ alone induced cell migration with an optimum at 2 mM for **fibroblasts** and at 10 mM for keratinocytes. Ca2+ alone at 2 mM only marginally enhanced **fibroblast** and keratinocyte migration. At higher concentrations Ca2+ suppressed the stimulatory Mg2+ effect. 2 mM Ca2+ combined with 2 mM Mg2+ showed an additive stimulatory effect on the migration of **fibroblasts** to fibronectin. These data suggest that extracellular divalent cations differentially influence the integrin-mediated cell migration. A concentration gradient of Mg2+/Ca2+, as reported in tissue injury, thus may play a regulatory role in cell migration required for tissue remodelling.

16/3,AB/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08437287 94364443 PMID: 7521847
Mg2+ and Ca2+ differentially regulate beta 1 integrin-mediated adhesion of dermal fibroblasts **and keratinocytes to various extracellular matrix proteins.**
Lange TS; Bielinsky AK; Kirchberg K; Bank I; Herrmann K; Krieg T; Scharffetter-Kochanek K
Department of Dermatology, University of Dusseldorf, Germany.
Experimental cell research (UNITED STATES) Sep 1994, 214 (1) p381-8, ISSN 0014-4827 Journal Code: EPB
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The specific requirements for divalent cations in the integrin-dependent adhesion and deadhesion of human dermal **fibroblasts** and human epidermal

keratinocytes to various extracellular matrix proteins have been studied in vitro. The adhesion of both cell types to collagen type I and to laminin was enhanced by Mg2+ in a concentration-dependent manner, while Ca2+ dose-dependently antagonized this effect, thus promoting deadhesion. The cation-dependent conversion between adhesion and deadhesion occurred already at 2 to 10 min after addition of the alternate cation and was almost completed at 20 min. Interestingly, Ca2+ could not reverse the Mg(2+)-enhanced adhesion of both cell types to fibronectin. Inhibition studies with function-blocking antibodies directed against distinct beta 1 integrins showed that the Mg(2+)-enhanced **fibroblast** adhesion to collagen type I was mediated by the alpha 1 beta 1 and the alpha 2 beta 1 integrins, whereas keratinocyte adhesion to collagen type I was mediated by the **alpha 2 beta 1 integrin** . Both cell types utilized the **alpha 2 beta 1** and the **alpha 6 beta 1** integrins for Mg(2+)-dependent adhesion to laminin and the **alpha 5 beta 1 integrin** for the adhesion to fibronectin. **Integrin** expression at the cell surface was not altered, indicating that divalent cation-dependent conformational changes of beta 1 integrins most likely regulate their functional activity.

16/3,AB/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07139567 93306622 PMID: 7686445
Alpha 6 integrin is up-regulated in step increments accompanying neoplastic transformation and tumorigenic conversion of human fibroblasts .

Lin CS; Zhang K; Kramer R
Laboratory of Cancer Cell Biology, Palo Alto Medical Foundation Research Institute, California 94301.
Cancer research (UNITED STATES) Jul 1 1993, 53 (13) p2950-3, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA49423, CA, NCI; CA51884, CA, NCI; DE10564, DE, NIDCR
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Integrins are a family of transmembrane glycoproteins that serve as cell-cell and cell-substratum adhesion molecules and help regulate cellular differentiation and proliferation. In malignant cells, which exhibit abnormal differentiation and growth properties, the expression of an altered integrin repertoire could therefore be expected. From a tumorigenic human fibrosarcoma cell line we isolated a unique complementary DNA corresponding to the alpha 6 integrin subunit. Northern blot analysis using this complementary DNA as probe indicated that alpha 6 integrin mRNA was abundantly expressed in all neoplastically transformed **fibroblast** cell lines but not in normal diploid **fibroblasts** . In addition to its potential as a marker for the neoplastic transformation of human **fibroblasts** , the alpha 6 integrin mRNA was also found to be consistently expressed at higher levels in tumorigenic **fibroblasts** than in immortalized but nontumorigenic **fibroblasts** . This differential expression of alpha 6 integrin was reflected at the cell surface protein level using cytofluorometric analysis with specific monoclonal antibody. In contrast, the levels of cell surface expression of other integrins were unchanged (such as alpha 3 and beta 1) or down-regulated (such as alpha 5) when transformed cells were compared with normal **fibroblasts** . The incremental up-regulation of alpha 6 integrin was selective and paralleled the progression of normal cells to immortalized cells and finally to tumorigenic cells. This elevated alpha 6 subunit associated with the beta 1 subunit to form a heterodimer receptor for laminin. Since fibrosarcoma cell invasion of basement membrane has been shown to involve **alpha 6 beta 1 integrin** , then the induction or

up-regulation of **alpha 6** expression is an important step in tumor progression and evolution to the invasive phenotype in fibrosarcoma.

16/3,AB/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07022887 93107190 PMID: 8416993
Integrins in point contacts mediate cell spreading: factors that regulate integrin accumulation in point contacts vs. focal contacts.
Tawil N; Wilson P; Carbonetto S
Center for Neuroscience Research, McGill University, Montreal General Hospital Research Institute, Quebec, Canada.
Journal of cell biology (UNITED STATES) Jan 1993, 120 (1) p261-71, ISSN 0021-9525 Journal Code: HMV
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
We have studied the function and distribution of the alpha **1** beta 1, alpha 5 beta 1 and alpha **6** **beta 1** heterodimers on type-**1** astrocytes with antibodies specific for **integrin** subunits (**alpha 1** , **alpha** 5, **alpha 6** , and **beta 1**). The **alpha 1 beta 1** heterodimer mediates adhesion to laminin and collagen, the alpha 5 beta 1 to fibronectin in an RGD-dependent manner. The alpha 5 beta 1 integrin is found in focal contacts in long-term cultures of well-spread astrocytes colocalizing with vinculin and the termini of actin stress fibers. alpha 1 beta 1 heterodimers can occasionally be found as small aggregates within focal contacts but they do not accumulate there. Instead, alpha 1 beta 1 integrins are found in punctate deposits called point contacts which are distributed over the upper and the lower cell surfaces whether laminin, collagen, fibronectin or polylysine is used as a substratum. Unlike focal contacts, point contacts contain clathrin but rarely codistribute with actin or vinculin. Two observations indicate that these point contacts are functional. First, mAb 3A3, directed against the rat alpha 1 subunit, inhibits the attachment of astrocytes to laminin and collagen. Second, during the spreading of astrocytes, a band of point contacts forms around the cell perimeter at a time when no focal contacts are visible. While alpha **1** **beta 1** integrins are found only in point contacts in astrocytes, the **alpha 6 beta 1 integrin** , another laminin receptor, is localized within focal contacts. Moreover, **alpha 1 beta 1** heterodimers accumulate in focal contacts in **fibroblasts** . Thus, the alpha subunit contributes, independent of its ligand, to functional integrin heterodimer accumulation in focal contacts or in point contacts. This accumulation varies among different cell types with apparently identical heterodimers as well as with the motile state (spreading vs. flattened) of the same cells.

16/3,AB/17 (Item 17 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06934864 93213708 PMID: 1363627
Expression of alpha 6 beta 1 integrin, **the laminin receptor, on subsets of normal murine lung** fibroblasts **and its upregulation by the inflammatory cytokines IFN-gamma and TNF-alpha.**
Felch ME; Willis RA; Penney DP; Keng PC; Phipps RP
Cancer Center, University of Rochester School of Medicine and Dentistry, NY 14642-8704.
Regional immunology (UNITED STATES) Nov-Dec 1992, 4 (6) p363-70, ISSN 0896-0623 Journal Code: AVT
Contract/Grant No.: CA-42739, CA, NCI; CA-55305, CA, NCI; HL-39949, HL,

NHLBI; +
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

The purpose of this investigation was to ascertain whether the alpha 6 integrin subunit was present on normal murine lung cells and **fibroblasts** , and if so, to determine the identity of the beta subunit coordinately expressed with alpha 6 and whether or not these integrin subunits could be regulated by cytokines. Previously, our laboratory isolated populations of Thy 1+ and Thy 1- **fibroblasts** from normal murine lung tissue. These cells differed in surface marker expression and in response to, and production of, pro-inflammatory cytokines. Research defining the properties of these two populations has led to the hypothesis that unique groups of **fibroblasts** exist within the murine lung. Though alpha 6 beta 1 is known to be expressed by platelets, lymphocytes, and epithelial cells, its presence and regulation on lung **fibroblast** subsets has not been explored. We now report the following findings: 1) the laminin receptor, alpha 6 beta 1, is present on 20-30% of freshly isolated normal murine lung cells in all three murine strains tested; 2) established Thy 1+ and Thy 1- murine lung **fibroblast** subsets and clones constitutively express alpha 6 beta 1 at varied levels; and 3) alpha 6 beta 1 expression on **fibroblast** lines and clones can be upregulated by treatment with IFN-gamma or TNF-alpha. Since these T cell and macrophage derived cytokines are known to be present during an inflammatory response, upregulation of alpha 6 beta 1 expression may facilitate recruitment and retention of lung **fibroblasts** in regions undergoing repair.(ABSTRACT TRUNCATED AT 250 WORDS)
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      Items   File
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         80     5: Biosis Previews(R)_1969-2001/Aug W2
          2     6: NTIS_1964-2001/Aug W4
          1    16: Gale Group PROMT(R)_1990-2001/Aug 15
         73    34: SciSearch(R) Cited Ref Sci_1990-2001/Aug W2
          9    35: Dissertation Abs Online_1861-2001/Jul
          1    44: Aquatic Sci&Fish Abs_1978-2001/Aug
          3    50: CAB Abstracts_1972-2001/Jul
   Examined  50 files
          3    65: Inside Conferences_1993-2001/Aug W2
         42    71: ELSEVIER BIOBASE_1994-2001/Aug W1
         50    73: EMBASE_1974-2001/Aug W2
         28    76: Life Sciences Collection_1982-2001/May
          1    77: Conference Papers Index_1973-2001/Jul
          5    88: Gale Group Business A.R.T.S._1976-2001/Aug 16
          1    94: JICST-EPlus_1985-2001/Jul W3
          4    98: General Sci Abs/Full-Text_1984-2001/Jul
          2   103: Energy SciTec_1974-2001/Jul B2
   Examined 100 files
         12   143: Biol. & Agric. Index_1983-2001/Jul
         19   144: Pascal_1973-2001/Aug W2
          2   149: TGG Health&Wellness DB(SM)_1976-2001/Aug W1
         61   155: MEDLINE(R)_1966-2001/Sep W2
          7   156: Toxline(R)_1965-2000/Dec
         58   159: Cancerlit_1975-2001/Jul
          4   172: EMBASE Alert_2001/Aug W2
          1   173: Adis LMS Drug Alerts_1983-2001/Jul W5
          1   194: CBD_1982/Dec-2001/Apr
   Examined 150 files
          4   266: FEDRIP_2001/Jul
          1   272: ONTAP(R) EMBASE_
   Examined 200 files
          1   342: Derwent Patents Citation Indx_1978-01/200143
          1   348: EUROPEAN PATENTS_1978-2001/AUG W01
         13   349: PCT Fulltext_1983-2001/UB=20010802, UT=20010726
          2   357: Derwent Biotechnology Abs_1982-2001/Sep B2
   Examined 250 files
         21   398: CHEMSEARCH(TM)_1957-2001/Jul
         57   399: CA SEARCH(R)_1967-2001/UD=13508
         17   420: UnCover_1988-2001/May 31
         96   440: Current Contents Search(R)_1990-2001/Aug W4
   Examined 300 files
          1   452: Drug Data Report_1992-2001/Jul
          2   459: Daily Essentials (Archival)_1996-2001/Jul W5
          3   484: Periodical Abs Plustext_1986-2001/Aug W2
   Examined 350 files
          1   636: Gale Group Newsletter DB(TM)_1987-2001/Aug 15
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   Examined 450 files
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S1	9	CYR61

1/3/1
DIALOG(R)File 35:Dissertation Abs Online

01755219 ORDER NO: AADAA-I9978767
Impaired placentation, compromised vascular integrity and embryonic lethality in Cyr61**-null mice**
Author: Mo, Fan-E
Degree: Ph.D.
Year: 2000
Corporate Source/Institution: University of Illinois at Chicago, Health Sciences Center (0806)
Source: VOLUME 61/07-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3399. 80 PAGES
ISBN: 0-599-84722-0

1/3/2
DIALOG(R)File 35:Dissertation Abs Online

01726802 ORDER NO: AADAA-I9951456
Identification and characterization of genes associated with mutant p53('val135)-induced cell transformation in Rat 6 fibroblast cell line
Author: Yam, Judy Wai Ping
Degree: Ph.D.
Year: 1999
Corporate Source/Institution: Hong Kong University of Science and Technology (People's Republic of China) (1223)
Source: VOLUME 60/11-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 5365. 219 PAGES

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01599325 ORDER NO: AAD98-01596
CLONING OF THE HUMAN HOMOLOG OF CYR61 **AND CHARACTERIZATION OF ITS BIOLOGICAL ACTIVITIES (IMMEDIATE EARLY GENE, HEPARIN BINDING, CELL ADHESION, VITRONECTIN)**
Author: KOLESNIKOVA, TATIANA VALERIEVNA
Degree: PH.D.
Year: 1997
Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 58/07-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3471. 92 PAGES

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01599324 ORDER NO: AAD98-01595
CYR61 **AND FISP12 ARE EXTRACELLULAR REGULATORY PROTEINS: ACTIVITIES AND MECHANISM OF ACTION (CELL MATRIX INTERACTIONS, CELL DIVISION, DIFFERENTIATION)**
Author: KIREEVA, MARIA LEONIDOVNA
Degree: PH.D.
Year: 1997

Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 58/07-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3617. 134 PAGES

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01592032 ORDER NO: AADNN-17206
ETUDE DE LA TRANSFORMATION ET DU CONTROLE DE LA PROLIFERATION CELLULAIRE PAR LES PROTEINES FOS (FRENCH TEXT, TUMOR SUPPRESSOR, FIBROBLASTS, CELL CYCLE CONTROL)
Author: AURELIO, BALSALOBRE
Degree: PH.D.
Year: 1996
Corporate Source/Institution: UNIVERSITE DE MONTREAL (CANADA) (0992)
Source: VOLUME 58/04-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 1681. 234 PAGES
ISBN: 0-612-17206-6

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01582729 ORDER NO: AADNN-17206
ETUDE DE LA TRANSFORMATION ET DU CONTROLE DE LA PROLIFERATION CELLULAIRE PAR LES PROTEINES FOS (FRENCH TEXT, TUMOR SUPPRESSOR, FIBROBLASTS, CELL CYCLE CONTROL)
Author: AURELIO, BALSALOBRE
Degree: PH.D.
Year: 1996
Corporate Source/Institution: UNIVERSITE DE MONTREAL (CANADA) (0992)
Source: VOLUME 58/04-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 1681. 234 PAGES
ISBN: 0-612-17206-6

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01375602 ORDER NO: AAD94-26517
REGULATION OF EXPRESSION OF GROWTH FACTOR-INDUCIBLE IMMEDIATE-EARLY GENES CYR61 **AND PIP92 (EMBRYOGENESIS)**
Author: LATINKIC, BRANKO VASILIJE
Degree: PH.D.
Year: 1994
Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 55/05-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 1749. 186 PAGES

1/3/8
DIALOG(R)File 35:Dissertation Abs Online

01327885 ORDER NO: AAD93-35171

BIOCHEMICAL AND FUNCTIONAL ANALYSIS OF CYR61, **THE PRODUCT OF A GROWTH FACTOR-INDUCIBLE IMMEDIATE EARLY GENE**
Author: YANG, GEORGE P.
Degree: PH.D.
Year: 1993
Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 54/08-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3953. 129 PAGES

1/3/9
DIALOG(R)File 35:Dissertation Abs Online

01257105 ORDER NO: AAD92-38021
CHARACTERIZATION AND EXPRESSION ANALYSIS OF THE GROWTH FACTOR-INDUCIBLE IMMEDIATE-EARLY GENE CYR61 **(** CYR61 **GENE)**
Author: O'BRIEN, TIMOTHY PAUL
Degree: PH.D.
Year: 1992
Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 53/08-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3911. 126 PAGES